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Toxic effects seen in a herd of beef cattle following exposure to ash residues contaminated by lead and mercury

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Abstract

Lead poisoning was diagnosed in three cattle along with increased mercury levels in the liver and kidney tissues of two of these animals. The clinical signs were different in each case and included salivation, anorexia, delayed menace response, delayed withdrawal reflex, head pressing, localized muscle fasciculation, reduced tongue tone, ataxia, rumen atony and seizures. Blood lead concentration was increased in all three cases to 0.76, 0.37 and 0.454 ppm. Post mortem changes characteristic of lead poisoning were only recognized in one case and included cerebro-cortical oedema, cortical neuronal necrosis and endothelial proliferation, especially at the tips of the cerebral gyri. The animals were poisoned by ingestion of lead-contaminated ash residues from a bonfire. The abnormal levels of mercury in the liver and kidney tissues of two animals may also be at least partly attributable to the intake of the metal in the ash residues. The levels of mercury in the three samples from the ash residue were relatively low (1.31, 0.7 and 2.1 ppm).

Keywords: Poisoning; Lead; Mercury; Ash residues; Seizures

1. Introduction

Among all livestock, lead toxicity remains one of the most common causes of poisoning. Sources of lead include lead-based paints, motor oil, discarded lead storage, batteries, machine grease, shotgun pellets, lead contaminated forages and lead for the wheel balance of tyres (Braun et al., 1997; Buck, 1975; Frape and Pringle, 1984; Schlerka et al., 2004; Sydler et al., 2001; Wunderlin et al., 1992). The use of lead in gasoline and paint has been reduced in recent years leading to a reduced risk of exposure to lead (Hoff et al., 1998).

In most animals the clinical signs of lead toxicosis relate to the central nervous system (CNS) and gastrointestinal systems (Baker, 1987; Cebra and Cebra, 2004). Lead poisoning in cattle can be acute, subacute or chronic, although the acute form occurs more frequently. Ozmen and Mor (2004) described a case of acute lead intoxication in six cows five days after the owner put them in an old battery factory. Clinical signs including violent movements, blindness, salivation, ataxia, and rapid and difficult breathing, increased heart rates, tremor, and coma were observed. In one of these cows, liver and kidney lead concentrations were 66.7 and 80.5 ppm, respectively. Concentrations in excess of 10 ppm are diagnostically significant for lead toxicosis in cattle (Baker, 1987). In the cases described by Schlerka et al. (2004), clinical signs of gastrointestinal involvement, such as diarrhoea, were observed along with

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nervous signs. Sydler et al. (2001) described a case of poisoning by chronic ingestion of lead in calves with a peracute clinical course. In this case the source of lead was a prime coat of white lead on old white painted doors. Wunderlin et al. (1992) documented a case of acute lead toxicosis in calves characterized by clinical symptoms including neurological and gastrointestinal symptoms. In this case, painted iron girders was the source of poisoning. The chronic form of lead poisoning is uncommon in cattle (Baker, 1987).

Sources of mercury for domestic animals include seed grains treated with organic mercurial fungicides, contamination of water by waste materials from certain manufacturing processes, such as paper production and accidentally contaminated feedstuffs (Boyd, 1985; Prior, 1976). In Western countries the use of seed grain treated with organic mercurial fungicides was banned more than 20 years ago making this an unlikely source of the mercury.

Acute poisoning with large amounts of inorganic mercurial compounds causes copious bloody diarrhoea, vomiting of bloodstained material and abdominal pain. Death occurs within a few hours owing to shock and dehydration. Depression, anorexia, emaciation, a stiff stilted gait which may progress to paresis, skin disorders, persistent diarrhoea, incoordination and convulsions have been reported in animals chronically exposed to low doses of inorganic mercury (Irving and Butler, 1975; Radostits et al., 2000).

Chronic organic mercurial poisoning causes neurological syndromes. Signs include abnormalities of gait, blindness, depression, muscle tremor and incoordination (Radostits et al., 2000). Boyd (1985) described an atypical outbreak caused by organomercuric poisoning. This outbreak was characterized by sudden death and multiple extensive haemorrhages in internal organs. Five calves poisoned with methylmercury (Ceresan L, E.I. duPont de Nemours & Company, Inc.) added to the milk, showed a chronic toxicosis characterized by sudden onset of nervous symptoms that rapidly developed into prostration and a moribund condition in 3–7 days (Herigstad et al., 1972).

2. Materials and methods

2.1. Preliminary report

In June 2003, four one-year old calves were found dead within a 10 day period on a beef cattle farm in the Austrian state of Lower Austria. The herd of 20 cattle was housed on a summer pasture. After the fourth calf died, three animals from the farm were transferred to the Clinic for Ruminants at Vienna's University School of Veterinary Medicine, because of an acute onset of neurological symptoms and one animal because of anorexia. The remaining animals were moved from the pasture into the stable.

The first case (Case 1) was a two-and-a-half year old male. This Limousin bull was referred with a one-day history of anorexia, along with signs of depression and lethargy. Case 2 was a 4-year-old Simmental cow referred because of rumen atony, salivation, forward locomotion, characterized by pushing the forelegs and head against the feeding trough, and a fever lasting one day. Case 3 was an 8-year-old Simmental-cross-Limousin cow that was admitted with a one-day history of anorexia. Cases 2 and 3 were admitted 11 days after Case 1 arrived at the Clinic for Ruminants.

2.2. Clinical study

A general physical and a neurological examination were performed on all cases. Clinical investigations were carried out following the criteria described by Baumgartner (2005a).

2.3. Laboratory investigations

Blood was collected at admission by jugular venepuncture for a complete blood count (Advai, Bayer) (Kraft, 2005), and a biochemical profile including alkaline phosphatase (AP), aspartate aminotransferase (AST), bilirubin (TBIL), creatine kinase (CK), glutamate dehydrogenase (GLDH), calcium (Ca), phosphorus (P) and iron (Fe) (Hitachi 911 Automatic Analyzer, Roche), (Kraft and Dürr, 2005). Blood gas analyses and potassium (K) were measured by Gem Premier Plus Autoanalysis (Instrumentation Laboratory) (Kraft and Wirth, 2005).

Cerebrospinal fluid (CSF) was collected by puncture of the subarachnoid space at the lumbosacral site in Case 2 and Case 3 after neurological assessment. Glucose, protein, and creatine kinase measurements were performed using a Hitachi 911 Automatic Analyzer (Roche) (Kraft and Dürr, 2005) and cell analyses were accomplished in a Fuchs-Rosenthal Cytometer (Fischer, 2005).

When lead toxicosis was suspected, blood samples were collected and submitted for blood lead analyses. Urine was collected in Case 1 within the timeframe when calcium versenate (CaEDTA), (Pharmacy Veterinary University Vienna, Austria) was administered at a dose of 73 mg/kg/day. Blood and urine lead analyses were performed using a graphite furnace atomic absorption spectrophotometer as described by Kenntner et al. (2001). The results of the blood lead analyses were available two to four days after the animals were admitted.

2.4. Post mortem examinations

A complete necropsy was performed in all of the three hospitalised cattle. All tissues were routinely processed for histopathology and stained with haematoxylin and eosin (H&E). Samples of the liver, kidneys and ruminal contents were collected during necropsy and submitted immediately for lead, cadmium and mercury analyses. The lead and cadmium levels were determined using a graphite furnace atomic absorption spectrophotometer, in which mercury was analysed by means of a mercury-hydride system (Kenntner et al., 2001).

For quality control a standard reference material was analysed. This was a sample of bovine liver 1577b from the US National Institute of Standards and Technology. The following certified concentrations of reference material were obtained for this reference material:

- lead: 0.129 ppm;
- cadmium: 0.50 ppm;
- mercury: 0.003 ppm.

Our analyses of this reference material gave the following results (mean of three analyses):

- lead: 0.134 ppm;
- cadmium: 0.514 ppm;
- mercury: 0.004 ppm.

3. Results

3.1. Clinical study, laboratory investigations and treatments

3.1.1. Case 1

Upon admission the bull was depressed and in sternal recumbency. The initial examination revealed a heart rate of 52 beats/min (normal reference range, 60–80 beats/min), respiratory rate of 16 breaths/min (normal reference range, 10–30 breaths/min) and a rectal temperature of 38.4 °C (normal reference range, 38.5–38.8 °C). Furthermore, the mucous membranes were noticeably pale, and salivation and anorexia were also present.

Neurological examination revealed a delayed menace response in both eyes as well as a delayed withdrawal reflex. The animal was barely responding to environmental stimuli. Acute toxicosis was suspected. The differential diagnosis included rabies, polioencephalomalacia and thromboembolic meningoencephalitis.

Haematological examination revealed a mild leucocytosis and a mild erythrocytosis (Table 1). The results of the serum chemistry and blood gas analyses are shown in Table 2. Results of the blood lead analysis were suggestive of lead poisoning (Table 3).

Treatment consisted of the IV administration of a 0.9% NaCl solution, 30% glucose solution (1 L 30% glucose per 10 L 0.9% NaCl solution), and thiamine (600 mg per 10 L 0.9% NaCl) at a constant rate infusion of 30 mL/kg/day for seven days. This regime is reported to decrease lead accumulation and relieve the toxic symptoms in cattle (Baker, 1987). Further treatment included the IV administration of oxytetracycline (10 mg/kg/day) for eight days and depot dexamethasone (0.02 mg/kg/twice weekly). Rumen fluid (10 L) and water (60–80 L) were administered orally for eight days. The lead chelator, calcium versenate (CaEDTA), (73 mg/kg/day) was administered by slow IV injection for four days after the diagnosis of lead poisoning was confirmed.

The bull showed a slight improvement from the toxicosis three days after the treatment with CaEDTA started. High lead concentrations were confirmed in the urine after the initial treatment with CaEDTA. Two hours after the beginning of treatment the urine lead concentration was 3.1 mg/L, rising to 13.76 mg/L 8 h later and averaging 19 mg/L on the following days of treatment.

The animal developed signs of respiratory disease (temperature 39.9 °C, laboured and irregular breathing, purulent bilateral nasal discharge, bronchial sounds on auscultation) by day 6. On day 7, the bull developed leucocytopaenia (1190 WBCs/μL) due to a neutrophilia (173.7 cells/μL) and a relative lymphocytosis (950.81 cells/μL). The animal was found dead eight days after admission.

3.1.2. Case 2

Physical examination at admission revealed depression, a heart rate of 120 beats/min, respiratory rate of 40 breaths/min, rectal temperature of 38.6 °C and reduced rumen motility. Upon neurological examination the animal showed head pressing, salivation, muscle fasciculation of the lips, reduced tongue tone, ataxia (grade, 3/5) and conscious proprioceptive deficits (most pronounced in the left fore- and hind-limbs).

Table 1
Haematological results

Parameters	Case 1	Case 2	Case 3	Reference range[a]
White blood cell count (n/μL)	11,480	12,960	10,650	6200–9500
Segmented neutrophils (%)	56.1	59.4	33.1	23–37
Lymphocytes (%)	35.6	34.0	51.8	53.0–67.0
Monocytes (%)	5.4	3.2	6.3	<4.0
Eosinophils (%)	1.4	2.5	7.6	1.0–7.0
Basophils (%)	0.6	0.5	0.7	<1.0
Red blood cells (10^6/μL)	8.26	6.48	6.77[b]	5.00–7.00
Haemoglobin (g/dL)	12.5	11.4	12.3	10.5–14.0
Haematocrit (%)	34	31.2	33.9	30–40
Mean corpuscular volume (fL)	41.2	48.2	50.1	40–60
Mean corpuscular hemoglobin (pg)	15.1	17.6	18.2	14–19
Mean corpuscular hemoglobin concentration (%)	36.8	36.5	36.3	26–34

[a] Baumgartner (2005c).
[b] Sporadic basophilic stippling.

Table 2
Clinical biochemistry and selected acid–base variables

Parameters	Case 1	Case 2	Case 3	Reference range[a]
Alkaline phosphatase (U/L)	71	32	34	<200
Aspartate aminotransferase	128	55	26	<30
Bilirubin (mg/dL)	0.38	0.15	0.25	<0.35
Creatine kinase (U/L)	1567	174	84	<30
Glutamate dehydrogenase (U/L)	9.54	19.65	3.73	<9
Calcium (mmol/L)	2.32	2.01	2.10	2.3–3
Phosphor (mmol/L)	1.62	2.61	2.82	1.6–2.3
Magnesium (mmol/L)	0.81	0.58	0.71	0.7–1.2
Iron (mmol/L)	39	51	53	27–40
Potassium (mmol/L)	3.05	2.88	2.72	4.4–5.7
pH	7.37	7.34	7.36	7.38–7.42
Acid–base excess (mmol/L)	0.3	−3.9	−3.1	1.0–6.0

[a] Baumgartner (2005c).

Table 3
Results of lead, mercury and cadmium analysis

	Case 1	Case 2	Case 3	Acute toxicosis
Lead[b]				
Blood (ppm)	0.76	0.37	0.454	>0.35
Liver (ppm)	1.501	11.155	37.67	>10
Kidney (ppm)	2.423	26.129	159.65	>10
Rumen contents (ppm)	4.08	14.13	–[a]	No reference
Mercury				
Liver (ppm)	0.873	0.750	<0.001	>100
Kidney (ppm)	0.612	1.031	<0.001	>10
Cadmium				
Liver (ppm)	0.056	0.161	0.764	No reference
Kidney (ppm)	0.131	0.995	0.001	No reference

References: lead (Baker, 1987); mercury-kidney (Buck, 1975); mercury-liver (Hapke, 1988).
[a] High portion of inorganic matter, which mainly consisted of lead salts.
[b] mg/kg tissue wet weight (ppm).

Results of the haematological examination revealed a leucocytosis. The red blood cell count was within the normal range (Table 1). Results of the blood gas analysis revealed acidosis, characterized by a venous blood pH of 7.34 and an acid base excess of −3.1 mmol/L. Serum biochemical abnormalities included increased AST (55 U/L), CK (174 U/L), GLDH (19.65 U/L), phosphorus (2.61 mmol/L) and iron (51 mmol/L). Moreover calcium (2.01 mmol/L) and potassium were lowered (2.88 mmol/L). All measured parameters are shown in Table 2.

Analysis of the CSF revealed a mild increase in the protein concentration (0.48 g/L; normal reference range <0.4 g/L), normal glucose (60 mg/dL; normal reference range 35–70 mg/dL) and increased creatine kinase (120 U/L; normal reference range 2–48 U/L). An increase in the cells (15 cells/μL; normal reference range 10 cells/μL) and in creatine kinase activity (120 U/L; normal reference range 2–48 U/L) were invalid because of the contamination of the CSF sample by blood.

Based on the herd history and clinical signs, a provisional diagnosis of acute lead poisoning was made. However, because of the poor prognosis and the financial constraints imposed by the owner, treatment was not attempted and euthanasia was performed.

3.1.3. Case 3

Physical examination at admission revealed the cow to be alert with a heart rate of 80 beats/min, respiratory rate of 22/min, and rectal temperature of 38.6 °C. Further physical examination findings included anorexia, mild bilateral conjunctivitis and no rumen sounds, whereas the results of thoracic and cardiac auscultation were normal.

Neurological examination revealed an abnormal movement of the tongue. There was normal resistance when withdrawing the tongue from the mouth, but an active obtrusion and an active dorsiflexion of the tongue was impossible. Swallowing was inadequate owing to pharyngeal dysmotility.

Haemogram abnormalities included mild leucocytosis (total WBC count of 10,650 cells/μL) and sporadic basophilic stippling of erythrocytes (Table 1). Serum biochemical analysis revealed increased creatine kinase and iron, hyperphosphataemia and hypocalcaemia (Table 2). Serum activities of all the hepatic enzymes were within the normal reference ranges (Table 2). Results of the blood gas analysis are shown in Table 2. Rumen fluid analysis revealed no abnormalities.

The CSF was clear and colourless, cell concentration (2 cells/μL), protein concentration (0.1 g/L) and creatine kinase activity (3 U/L) were within normal range.

The clinical signs at the initial evaluation were not diagnostic for lead poisoning. However, based on the herd history lead poisoning was a major differential diagnosis in this animal and blood was submitted for lead analysis.

Supportive care was given until the results of the blood lead analysis were available. This included maintenance fluid therapy (1 L 30% glucose per 10 L 0.9% NaCl solution and thiamine 600 mg per 10 L 0.9% NaCl solution) to prevent hypoglycaemia and dehydration. During the following two days the cow appeared more distressed and no improvement of the anorexia was recognised.

Over the following days the cow showed increasing signs of lethargy and depression and episodes of seizure-like

activity induced by loud noisiness and passive dorsiflexion of the neck. As time progressed spontaneous general seizures were also seen. During the seizure the animal became recumbent, together with opisthotonus and a retrocollis to the left side. Additionally, a bilateral horizontal nystagmus, ventromedial strabismus, and paddling movements of the limbs and focal periodic contractions of truncal and facial muscles were recognised (Fig. 1).

The seizures were characterized by apnoea of 30 s. An electrocardiogram was performed during the seizure according to Baumgartner (2005b). Atrial fibrillation with an atrial frequency of approximately 300 bpm was evident throughout the recording period. At the beginning of the apnoea episode the ventricular frequency increased immediately from 90 to 132 bpm for 10 s, decreasing to 60 bpm thereafter with constant, physiological QRS-complex morphology but irregular rhythm. At the end of the seizures the animal was able to get up without assistance.

As the number of seizures progressed, the duration of the seizures increased from about 1–3 min. In the time between the seizures the animal was depressed. The suspected diagnosis of lead poisoning was confirmed by the results of the blood lead analysis (Table 3).

The cow was euthanased at the owner's request, because of the poor prognosis and the cost of further treatment.

3.2. Pathology

3.2.1. Case 1

Necropsy revealed a large amount of small blue-grey particles in the forestomachs, focal greyish discolouration of the intestinal mucosa as well as severe croupous pneumonia, fibrinous pleuritis of the cranioventral portions of the lungs, and extensive subendocardial haemorrhages. Histologically, a slight interstitial nephritis and discrete areas of lung oedema were found. Sections of the cerebrum, cerebellum, brain stem and liver were histologically unremarkable.

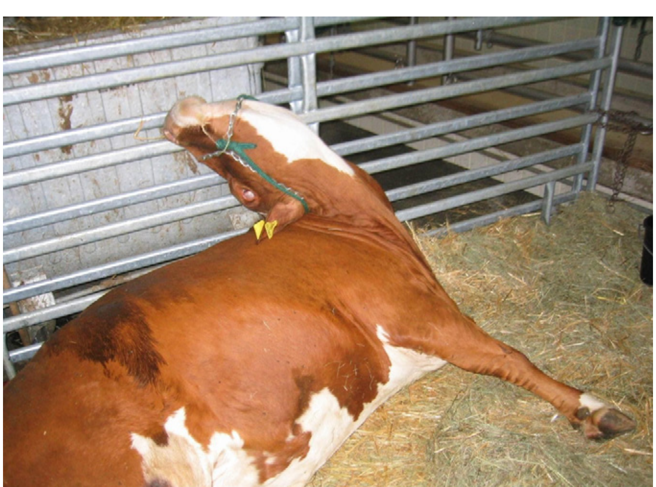

Fig. 1. Seizure of Case 3: note the ventromedial strabismus and the retrocollis to the left side.

3.2.2. Case 2

At necropsy small blue-grey particles were found in the reticulum. Microscopic renal lesions of mild interstitial nephritis and membranous glomerulonephritis were also seen. Hyperaemia was diagnosed in all parenchymal organs. Moreover, a mild focal non-suppurative leptomeningitis was observed.

3.2.3. Case 3

At necropsy, abscesses in the liver were found. Reticular contents consisted of roughage mixed with fluid and large amounts of lead containing blue-grey particles nearly 1 cm in diameter. Apart from the significant congestion of the kidneys and spleen, no substantial gross lesions were evident in the lungs, heart or kidneys. Microscopic CNS lesions consisted of cerebral cortical oedema, cortical neuronal necrosis, endothelial proliferation, especially at the tips of the cerebral gyri, and a focal suppurative leptomeningitis; furthermore the neuronal necrosis in the spinal cord was found. With the exception of a chronic pericholangitis and myocardial sarcosporidiosis, no notable extraneural microscopic lesions were found.

3.3. Lead, cadmium and mercury analyses of tissue samples and ruminal contents

3.3.1. Case 1

Lead concentrations in the liver and kidneys were not diagnostic for lead poisoning. The lead concentration in the ruminal contents was 4.08 ppm. The concentrations of cadmium in the liver and kidneys were considered to be normal – 0.056 ppm (tissue wet weight liver) and 0.131 ppm (tissue wet weight kidney). The mercury levels were higher than the official tolerance level of 0.1 ppm for bovine tissue in Austria. The kidney contained 0.612 ppm (tissue wet weight), and the liver contained 0.873 ppm (tissue wet weight) mercury (Table 3).

3.3.2. Case 2

In this case the diagnosis of lead poisoning was supported by tissue and blood lead concentrations that showed values higher than accepted norms (Table 3). The concentrations of cadmium in the liver (0.161 ppm tissue wet weight) and kidneys (0.995 ppm tissue wet weight) were within the normal range. The mercury level in the liver and kidneys was up to 10 times higher than the official tolerance level in bovine tissues (0.75 and 1.031 ppm) (Table 3).

3.3.3. Case 3

Lead concentrations in the blood, liver and kidney samples were much higher than normal values. In the rumen a very high portion of inorganic matter (75.9% dry matter) was found. Gravimetric analyses (as lead chromate and lead chloride) proved that the inorganic part in the rumen consisted mainly of lead salts, demonstrating the uptake of a very high amount of inorganic lead (Table 3).

Cadmium concentrations in the liver and kidneys were considered to be normal (<0.001 ppm tissue wet weight). Mercury analysis performed on the liver and kidney samples revealed concentrations lower than 0.001 ppm (Table 3).

3.4. Pasture

Inspection of the pasture was carried out by the owner on the same day that Case 1 was admitted to the Clinic for Ruminants. He did not find the source of poisoning on the first inspection. However, inspection on the next day revealed ash residues from an illegal bonfire, scattered over an area with a radius of 15 m. The small blue-grey particles found in the rumen where also found in the ash-residues. These consisted in large part of solidified lead metal. The lead, cadmium and mercury content of the ash-residues including the blue particles were of 999, 0.18 and 2.1 ppm, respectively. Moreover, in a second ash-sample, lead concentration was 700 ppm, cadmium concentration was 1.31 ppm and mercury concentration was 1.24 ppm. The ash residue was probably formed by the combustion of lead and mercury particles from battery plates, paint chips or other sources.

4. Discussion

The unusual nature of this outbreak is of interest as in all three cases there were different clinical symptoms at the time of admission and different results at necropsy. O'Hara et al. (1995) documented a heifer with lead toxicosis treated with $CaNa_2EDTA$ for six days. The heifer showed a slight improvement from the plumbism but developed signs of respiratory disease.

The course of the disease in Case 1 was similar in many ways to the case described by O'Hara et al. (1995). Liver and kidney lead concentrations in the case described by O'Hara et al. (1995) were measured after euthanasia on day 6 5.75 and 8.43 ppm, respectively. In contrast to this, the lead in the liver and kidneys was measured post mortem were much lower.

Liver and kidney mercury levels in Case 1 were 0.873 and 0.612 ppm, respectively. The normal background level of mercury in bovine livers and kidneys was described by Prior (1976) as 0.0007 and 0.008 ppm, respectively. In Austria, the official tolerance level in bovine tissue is 0.1 ppm (Austrian Ministry for Health, 1997). In acute toxicosis, mercury residues in the kidney exceed 10–15 ppm (Buck, 1975). The liver and kidney mercury level in Case 1 was higher than the official tolerance level in Austria (Austrian Ministry for Health, 1997), but much lower than those described for acute toxicosis. However, increased mercury levels in bovine tissues in Austria have not been found in recent years. The cause of the elevated mercury concentrations in the organs of two of the affected bovines could be the ingestion of some mercury-containing ash. However, the involvement of mercury in the toxic syndromes described here is a matter of speculation. The possible synergism between lead and mercury in cattle is not described in the literature.

According to Baker (1987) histopathological brain lesions are usually observed in acute, subacute and chronic lead poisoning. Why this was not seen in Case 1 is a matter of speculation. The authors assume that the beginning of the CaEDTA treatment was at an early stage and prevented a lot of capillary damage. Subendocardial and subepicardial haemorrhages have been described in the literature resulting from mercury poisoning in cattle (Irving and Butler, 1975; Boyd, 1985; Simpson et al., 1997). In the case documented here extensive haemorrhages were recognised in the endocardium. However, lead poisoning can also cause acute endothelial injury that leads to multiple haemorrhages within the lung, alimentary tract and heart.

In chronic lead poisoning normocytic and normochromic anaemia and basophilic stippling of the erythrocytes may develop. These haematological changes however are not considered to be a diagnostic characteristic in ruminants (Baker, 1987; Cebra and Cebra, 2004). In the cases described here basophilic stippling of the erythrocytes was only seen in Case 3. The shape and size of the abnormalities of erythrocytes were not seen in any of the cases.

Case 2 had high levels of mercury in the liver and kidneys (1.031 and 0.75 ppm, respectively) as was seen in Case 1. In contrast to the blood lead and tissue lead concentrations, necropsy findings were not diagnostic for lead poisoning in Case 2. The clinical symptoms of CNS involvement were pronounced at admission in Case 2. This finding is in contrast to Case 3. As in Case 1, prominent histological changes in the CNS indicating lead poisoning were not found in Case 2. The acute form of lead poisoning has a short course (12–24 h) and the animals may be found dead without prior observation of clinical signs. In these cases, lesions seen post mortem are often not distinctive (Cebra and Cebra, 2004). In Case 2 the brain lesions described in cattle (Baker, 1987) with a subacute or chronic form of lead poisoning were not seen.

The clinical course of the disease in Case 3 did not suggest lead as the main cause of poisoning. Brief reports in the literature described the possibility of convulsive seizures in cattle with chronic forms of lead poisoning. However, the course of these seizures has not been described in detail. In Case 3, seizures combined with apnoea and a sudden rise or drop of heart rate led to the conclusion of vegetative dysfunction due to seizure activity. In contrast to Cases 1 and 2, in Case 3 the brain lesions were similar to the lesions described by Buck (1975) and Baker (1987) in cattle poisoned with lead.

Lead poisoning damages red blood cells, the kidneys, bone and CNS. There is considerable variation in the extent and nature of the lesions resulting. The cause of this variability is not clear but the duration of exposure is likely to play a key role (Ozmen and Mor, 2004).

In conclusion, clinical and histological findings in the cases described here were not typical of lead poisoning.

The concentration of mercury in the ash-samples was low, therefore the involvement of this metal in the observed cases remains presumptive at best.

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